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SPECIAL ARTICLE

THE PRESENT STATUS OF ELECTROCARDIOGRAPHY IN THE DIAGNOSIS, PROGNOSIS AND TREATMENT OF HEART DISEASE

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HISTORICAL.

The normal electrocardiogram.

*Value of electrocardiogram in diagnosis.
Some of the abuses of the method.*

*Recognition of disordered mechanisms of
the heart without the aid of graphic methods
is possible in most instances.*

*Conditions which are shown only by the
electrocardiographic method.*

*Value in prognosis and treatment must
await further study and experimental work.*

*The ultimate goal is to learn what the in-
strument may teach us and then to dispense
with its use for routine work.*

THE credit for the introduction of the string galvanometer into the field of clinical medicine should be given to Einthoven. Long before his time Matteucci (1843) had discovered that electromotive force emanated from the heart itself. This was confirmed in 1849 by DuBois Reymond by the use of a very delicate galvanometer. Further confirmation came independently by Kölliker and Remak in 1850. Müller and Kölliker showed later (1850-1856) that a special current was developed in the auricles during their contraction. The invention of the capillary electrometer by Lippmann in 1873 led to the construction of the hypersensitive capillary electrometer. Waller, in 1887, suggested that there was a ratio between the contracting force of certain heart muscles and the current that emanates from them. The Ader registration machine or recorder, described in 1887 and used for submarine cable work, consisted of a long vertical wire stretched in a magnetic field.

Current was passed through the wire, which was drawn from side to side by the poles of the magnet, while a ray of light from a lamp threw a shadow of a minute portion of the moving wire on a moving strip of photographic paper in the form of an undulating dotted line. Einthoven's invention is an adaptation of this instrument to the study of heart disease, and, as originally constructed, has remained the standard instrument for clinical work.

Einthoven's instrument makes use of a delicate silvered quartz filament or fiber which can scarcely be seen by the unaided eye. It is suspended between the poles of a powerful electro-magnet. Any current passing through the conducting filament, which is placed in a magnetic field at right angles to the magnetic current, causes the filament to oscillate forward and backward according to the ascent or descent of the current. The amount of oscillation depends upon the strength of the magnetic field, the strength of the action current and the resistance in the filament. The movements of the filament, activated by cardiac action, are recorded by projecting its shadow, magnified by a high-power microscope, on a moving photographic plate or film. The time interval is recorded by photographing the shadow of a rotating spoked wheel activated by a tuning fork. The electromagnets are fed by an accumulator battery. The source of light was originally provided by a powerful arc lamp with a water chamber interposed to prevent injury to the filament. Many clinicians today employ an incandescent lamp (Point-O-Lite), which gives a constant beam of brilliant light. The action current originates at the heart and is drawn off by non-polarizable electrodes, placed on convenient and suitable parts of the body. Several types have been devised, but the one described by Cohn, and perhaps the most suitable, consists of a sheet of lead foil 7.5 cm. wide by 22 cm. long and covered by a strip of rubber sheet 9 cm. wide by 30 cm. long. The strips are fastened together near one end by a brass screw which carries the binding post. Warm salt solution is used as the electrolyte. The electrode is applied over gauze soaked in the solution after preparing the skin, to provide satisfactory contact. The current passing through the filament represents, not only the cardiac action currents, but also the difference of potential between the parts of the body to which the electrodes are applied.

In the body there are action currents of other tissues besides the heart muscle which produce variations in the potential. To offset this, a battery current is introduced which neutralizes this so-called skin current. The tension on the filament is adjusted so that each millivolt of current causes an excursion of one centimeter on the plate. This is the Einthoven standard for comparison, which is generally used in clinical work.

Briefly, then, the complete apparatus consists of (1) a string galvanometer, including an achromatic

microscope; (2) a constant source of brilliant light; (3) non-polarizable electrodes; (4) an accumulator battery for the magnetic field; (5) an apparatus for testing the susceptibility of the galvanometer; (6) an apparatus for compensating the "skin current"; (7) a photo-registering apparatus; (8) time-marking apparatus.

Electrodes are usually applied at three points, namely, right and left wrists and left calf. By a combination of these in pairs, we derive the three leads which are related to the action currents in different parts of the heart muscle, and the differences in potential established between the connections at the extremities. Thus:

Right arm and left arm constitute lead I.

Right arm and left leg constitute lead II.

Left arm and left leg constitute lead III.

From these leads the direction, amplitude, and time of the cardiac action currents can be expressed graphically, and a study of these constitute *electrocardiography*.

While the apparatus and its operation appears complicated in description, little difficulty is encountered in acquiring the necessary technical skill for manipulation. Greater skill and study are necessary in interpreting the three curves or leads which constitute the *electrocardiogram*. The *normal electrocardiogram* consists of a series of deflections which are associated with the events of a cardiac cycle, beginning with the origin of the impulse at the sino-auricular node and traversing the auricle, auriculo-ventricular node, ventricular conduction system and ventricular muscle and resulting in contraction. The deflections apparently result from electrical changes accompanying conduction of the impulse and contraction of the muscle. The deflections or waves are grouped according to their occurrence in the cycle; thus P waves are associated with auricular activity and the Q, R, S, and T waves accompany ventricular activity. The electrical changes in the heart precede the actual contraction of the muscle by a brief period. It is not necessary to go into the controversies which were waged about the causation of the different deflections in the normal cardiac cycle. Experimental work is helping to clear up many of the debated points. The final phase, known as the T wave, is the least well-understood deflection of the normal electrocardiogram. The original work of Einthoven (1900-1907), the subsequent studies of Frank and Hess, Eppinger and Rothberger, Kahn, Hoffman, Barker, Kraus and Nicolai, Eyster and Meek, and the brilliant researches of Lewis and his associates, laid the foundations for our present knowledge of the electrocardiogram in health and disease. Since 1910 the literature dealing with electrocardiographic studies has assumed voluminous proportions. A list of the more important contributions is appended. The recent studies have dealt largely with the applications of electrocardiography in diagnosis, prognosis, and treatment.

There can be no question but that the introduction of the electrocardiograph into experimental and clinical medicine has cleared up much of the confusion about the heart. These correlated studies shall serve for all time as one of the most brilliant applications of experimental physiology to clinical

medicine. The cardiac irregularities, one by one, have been studied and described. Heart-block, which was fairly well understood by experimental studies and clinical observations with the polygraph, has been further clarified by electrocardiographic work. This is particularly true of blocking of impulses or defective conduction in the bundle of His, its branches and its arborizations in the ventricles. In the course of extensive studies, the limitations of assistance which we may secure from the electrocardiographic method have been delineated. There are patients who are suffering from obvious cardiac failure who show no deviations from the normal so far as the electrocardiogram is concerned. Patients with extensive valvular disease may show normal electrocardiograms. Some of the more severe cardiac manifestations, such as pulsus alternans, which are demonstrable by the polygraphic or spynomanometric methods are shown only rarely on the electrocardiogram. However, in such cases the T waves frequently show abnormalities which have come to be associated with grave cardiac disorders.

VALUE IN DIAGNOSIS

At the outset it should be stated that one who is skilled in electrocardiography and has carefully studied the associated clinical manifestations of disordered action of the heart seldom needs to make use of the electrocardiographic method in diagnosis. This has been made possible by the brilliant observations of Mackenzie and others with the polygraph, and extensive studies by Lewis and numerous physiologists and clinicians with the electrocardiograph. Such a state of affairs does not necessarily condemn the electrocardiographic method as having no place in clinical medicine. Nor does it mean that such an instrument should be restricted to the research laboratories and the medical schools. The ultimate goal in the use of all instruments of precision, as Mackenzie and others have maintained, is to dispense with their use when we have learned all that they may teach us. This stage, however, has not been reached with the electrocardiograph in diagnosis and less so in prognosis and treatment.

Practically all Class A medical schools are today providing some training in the interpretation of electrocardiograms. An increasing amount of instruction is being given in the methods by which disordered mechanisms may be recognized, without instrumental aid. This is highly desirable for those who are entering general practice and may not be able, for many reasons, to carry on their instruction in graphic methods at the bedside. The polygraph, which is portable, may replace the electrocardiograph to some extent, and should be a part of the armamentarium of those doing internal medicine. Some provision must be made for those physicians who have not been instructed in instrumental methods or the interpretation of the findings and the clinical symptoms and signs which they present in the patient. A considerable proportion of patients who are sent for electrocardiographic study present normal variations or simple irregularities which could be readily recognized by the clinician. The patients with such disorders should be spared the expense and the fear that they are suffering from heart disease.

At times the reassurance that may come from the graphic registration and demonstration of a normal or inconsequential finding may be of value in dealing with certain types of patients, but this is seldom necessary for the able clinician.

Experience has repeatedly shown that clinicians should seldom rely on laboratory findings for the diagnosis in any given case. This is also true of the results which may be obtained with the so-called instruments of precision. In rare instances instrumental methods may reveal unsuspected conditions. In general, however, there are symptoms or signs which reveal to the careful observer the true nature of the disorder. Although electrocardiography has helped to place our present knowledge of cardiac disease on a firmer basis, we should not lean too heavily on the results it may show. There are times when we may be misled by normal electrocardiographic findings when the patient is shown to have obvious heart failure. On the other hand, we may err in assigning a grave prognosis in a case where the electrocardiogram shows extensive damage of the heart. It should be remembered that the prognostic significance of certain abnormalities is drawn from average experience, based on statistics subject to wide individual variations.

There are certain normal variations of the electrocardiogram which must be kept in mind. The shape of the curves in the three leads is constant for the individual for long periods of time, barring disease, and may, therefore, be of value in identification. The shape complexes may vary with respiration or changes in position of the heart, produced by displacements, etc. In the case of complete transposition of the viscera, the complexes in Lead I are inverted. None of these call for electrocardiographic confirmation, although they must be borne in mind in interpreting the graphic records.

Inasmuch as the ultimate goal in medicine is to dispense with instrumental aid, when we have learned all that it may be able to teach us, it would be advisable to attempt an exposition of the conditions where such aid is and is not necessary. In the paragraphs which follow, an effort will be made to show the present status of electrocardiography as an asset in clinical medicine. Simple methods of detecting disorders of the heart which require only sight, hearing and touch will be suggested.

The *irregularities* of the heart often confound the physician. The commonest of these is *sinus arrhythmia*, which is of frequent occurrence in children and young adults. Usually the variation in rhythm is associated with respiration, with an increase of rate during inspiration and a slowing during expiration. A deep breath, held, will often cause a marked slowing of the heart. Exercise or anything which speeds up the heart tends to eliminate the irregularity. A study of the jugular pulse in the neck usually shows the three normal waves. This irregularity is of no clinical significance, can usually be differentiated without instrumental means, and yet is frequently mistaken for a serious disorder. Extrasystoles may be associated and suggest auricular fibrillation. *Sino-auricular tachycardia* is a simple tachycardia associated with exercise, febrile and toxic conditions, and is usually regarded in its proper light. *Sino-auricular*

bradycardia with a rate of 50-60 may be a normal finding to confuse with heart-block. A study of the jugular pulse and a general examination of the patient will usually make the diagnosis. *Extrasystoles*, ectopic beats, or premature beats are also common, especially in young people, and usually have no great significance. They are often attributed to toxic influences or irritations arising outside the heart. In persons past middle life they are often associated with more general cardiac disease, and when arising from multiple foci, or when followed by alternation of the pulse, have more serious significance. When associated with muscle damage they may be forerunners of the tachycardias, such as auricular fibrillation, auricular flutter, or others. Extrasystoles arising in the auricles usually alter the rhythm of the heart, the new rhythm taking its time from the time of the inception of the extrasystole. Those arising in the ventricle usually do not disturb the underlying rhythm which takes its time interval from the last normal beat preceding the extrasystole. Extrasystoles may rarely be interpolated; that is, may fall between two normal beats without altering the rhythm. By listening to the heart, the extrasystole can usually be detected, which may not give rise to a perceptible pulsation at the wrist. This should be differentiated from dropped beats which belong properly under heart-block. By carrying the time with the foot, the changes in the underlying rhythm may be studied. By watching the jugular pulse in the neck, the presence or absence of the "A" wave may help to differentiate an auricular from a ventricular extrasystole. Extrasystoles frequently come in a definite sequence; that is, every second, third, fourth beat, and so on. If so, we may have a pulse at the wrist suggesting alternation, a bigeminal pulse, a trigeminal pulse, and so on. Exercise or anything which will increase the rate of the heart usually causes extrasystoles to disappear and give way temporarily to a regular rhythm. If the myocardium is severely damaged, extrasystoles may be increased by exercise. Electrocardiograms are of value in localizing the point of origin of extrasystoles and, when arising from multiple foci, would speak for grave disturbances. Multiple ventricular extrasystoles, associated with defective conductivity, may be forerunners of ventricular tachycardia and ventricular fibrillation, and indicate a bad prognosis. Chloroform anesthesia would be contra-indicated. Adrenalin should not be administered alone or in conjunction with chloroform anesthesia. By a careful study of the patient, the type and seriousness of extrasystoles can usually be determined. *Nodal rhythm*, where the impulse arises at the auriculo-ventricular node and passes in both directions, causes marked disturbance in the cardiac mechanism. Such a rhythm may be detected by a study of the jugular pulse. The rhythm is usually regular and rapid, but may be slow. The "A" wave in the jugular pulse may be present, coming close to the "C" wave, or may be submerged in it. The electrocardiogram gives a clear interpretation of this disorder, showing the site of origin of the impulse in the junctional tissues.

One of the commonest irregularities is *auricular fibrillation*. An electrocardiogram is seldom required to diagnose this condition. The absolutely irregular

rhythm, variation in force and volume, generally rapid rate (90-160), deficit between apex and pulse rate, absence of regular "A" waves in the jugular pulse, increase of the irregularity and breathlessness with exercise, and response to digitalis or quinidine differentiate this irregularity from others. A fibrillation, where partial or complete block is present, may be confusing. An auricular flutter, if showing an irregular ventricular rhythm, may simulate auricular fibrillation, but usually an underlying rhythm will be detected. Auricular fibrillation with slow undulating fibrillary auricular waves in the jugular pulse may suggest auricular flutter. The electrocardiogram is an aid in diagnosing these borderline conditions and in assessing the associated myocardial damage when taken in conjunction with the general clinical picture. With auricular fibrillation there is usually associated valvular or degenerative disease or toxic conditions, such as thyroid disease.

Auricular flutter, which is closely allied to auricular fibrillation, usually has a ventricular rate of 90 to 150, but may be above or below this rate. The rhythm is usually regular, but may be irregular. The auricles are usually beating about 300 times per minute and the ventricles fail to respond to each second, third or fourth beat because some of the ectopic impulses are blocked after passing through the auricles. The clinical symptoms are usually pronounced. The heart-sounds suggest a recurring irregularity or may be regular. The jugular pulse shows rapid regular "A" waves at about 300 per minute, with superimposed "C" and "V" waves. Vagal pressure usually causes a marked slowing of the ventricular rate when the "A" waves in the opposite jugular may be clearly noted. Exercise may increase the block and slow the ventricular rate. Quinine derivatives apparently have little effect on auricular flutter, but ample doses of digitalis may bring about auricular fibrillation which may then change to a regular rhythm if digitalis is withdrawn. Electrocardiograms are useful in exact diagnosis of the irregularity and in estimating the general muscle damage. Mitral disease and degenerative myocardial diseases are frequently associated. *Auricular tachycardia*, which is usually paroxysmal in type, generally shows a rate of 160-280 per minute. The impulses arising outside the pacemaker at the sinus take the center of the stage for a time and the heart beats rapidly and regularly. The clinical symptoms are generally marked, leading to anxiety on the part of the patient, family, and physician. Nothing can be determined at the heart except the rapid rate and regular rhythm. The jugular veins are distended and distinct waves are not easily made out. Anything which stimulates the vagus nerves may terminate the attack, in which case the regular, normal rhythm may be resumed in a spectacular manner. Quinidine by mouth or quinine intravenously may cause prompt cessation of the attack, which in itself is rarely fatal.

Any one of the disorders, namely, auricular fibrillation, auricular flutter and auricular tachycardia, may be paroxysmal in nature and, as such, probably constitute many attacks of *true palpitation* described by patients. Inasmuch as the attacks terminate promptly in a great majority of cases, clinicians

should be able to differentiate them without the aid of instruments because of the divergent prognostic and therapeutic features. Electrocardiograms show the location of the abnormal pacemaker in the latter two conditions.

Ventricular tachycardia is less common than auricular tachycardia, usually paroxysmal, and is a grave condition, occurring more often in extensive myocardial disease such as seen with coronary occlusion. The disordered rhythm with rapid rate and general condition of the patient leave little doubt in the mind of the clinician of the seriousness of the patient's malady. This condition probably precedes *ventricular fibrillation* which, if permanent, is not compatible with life. In both of these conditions, electrocardiograms are of value in instruction to complete the list of possibilities.

Heart-block may be noted in a variety of forms. *Sino-auricular* block is comparatively rare, where overstimulation of the vagus may cause the entire heart to stand still. Upon listening to the heart, no sounds would be heard and there would be no pulsations in the neck during the pause. This form should be differentiated from complete auriculo-ventricular block where the auricles continue to beat. Electrocardiograms may be necessary to aid in the decision. *Delayed conduction time* usually precedes the more severe forms of heart-block. The impulse from the pacemaker is delayed in its course from the auricle to the ventricles. A study of the jugular pulse will usually show that the "A" wave precedes the "C" wave by an interval greater than normal. Pressure on the vagus may increase the block. In many cases there are also dropped beats, where the ventricle fails to respond to an impulse coming from the auricles. The pause, indicating absence of ventricular contractions, may be detected by auscultation at the apex and noted in the jugular pulse. The pauses may occur rhythmically, tending to confuse with extrasystoles. An electrocardiogram is seldom necessary to differentiate, but is of value in showing the associated changes in the ventricular muscle. *Complete heart-block* may be permanent or paroxysmal in nature, as is seen in Adams-Stokes syndrome. The ventricles are usually beating at a rate of 20-50, but may show wider variations. The auricles are usually regular, but may show auricular fibrillation or flutter or other irregularities. There is no association between the rhythms in the upper and lower chambers of the heart. If the ventricular rate is slow, the auricles may be heard beating at a more rapid rate at the base of the heart. The jugular pulse usually shows the independent rhythm of the auricles; and there is usually evidence of degenerative disease in the heart. Syphilis, acute infections, and congenital lesions may be concomitant. By a careful study of the clinical findings, instrumental aids are not often necessary to decide as to the existence of complete heart-block. The original descriptions of Stokes and Adams are examples of the value of careful clinical observation. The blocking of impulses, which may occur in the bundles of His and its branches, and arborizations produce electrocardiograms which are, as yet, the least understood of all the abnormal mechanisms. The lesions which bring about such changes in the conductivity and contrac-

tility of heart muscles are often diffuse and variable, and the electrocardiograms produced thereby differ from characteristic curves shown by experimental study. We may not be able to demonstrate histologically the lesion which produces a given abnormality in the electrocardiogram. Clinically, we are not able to differentiate between a blocked impulse in the right or left branch of the bundle of His, nor are we able to state definitely that there is widespread interference with the impulse around the papillary muscles or beyond in the ventricular walls. The electrocardiographic method is invaluable in estimating the significance of some of these grave disturbances. The interference of the conduction of the impulse in the ventricles will be shown. The shape and duration of the "Q, R, S" complex and the shape and deviation of the "T" wave will give us an idea of the extent of damage. The general condition of the patient may suggest a grave cardiac condition. The poor quality of the first sound or the split first sound at the apex of the heart may be the only abnormality noted on general examination. Pulsus alternans may be noted when taking systolic blood-pressure readings and rarely by feeling the radial pulse in marked cases. Polygraphic tracings may reveal constant alternation or only after extrasystoles in such cases, and would be a help in diagnosis and prognosis.

The nature of *pulsus alternans* is not fully understood, but of its seriousness there can be no doubt. This is one condition where the electrocardiographic method gives us very little help, unless we can show changes in the ventricular complex which, in themselves, are known to be of equal seriousness and significance.

In *irritable heart*, strong respiratory cyclic oscillation of the electrical axis with sinus arrhythmia may be of assistance in making the diagnosis.

Relative *hypertrophy* or *preponderance* of one side of the heart over the other may be shown by the electrocardiographic method. The findings may be of value in a study of congenital or valvular heart disease, hypertension, and nephritis, but should be used only in conjunction with the general clinical and roentgenographic findings. Changes in the "P" waves may indicate unequal hypertrophy of the auricles.

In considering the disordered mechanisms of the heart from a diagnostic standpoint, it should be kept in mind that combinations may occur which make accurate diagnosis almost impossible without the assistance which the electrocardiograph may render.

VALUE IN PROGNOSIS

The electrocardiographic method has proven of great value in prognosis. This is largely due to the fact that recognition and classification of disordered mechanisms by this method enabled us to study the various groups over a period of years. Apparently, all the possible cardiac disturbances have been observed experimentally and clinically. As a general rule, individuals who show multiple disturbances in the normal cardiac mechanism, as shown electrocardiographically, have a grave prognosis. Those who show simple extrasystoles or sinus arrhythmia

have an excellent prognosis. Paroxysmal auricular tachycardia is usually not of a serious nature. Auricular fibrillation is often paroxysmal and not serious, but when permanent is usually attended by increasing disability because of the frequent association with a failing heart muscle. Auricular flutter parallels auricular fibrillation. The finding of bundle-branch block or arborization block, which may be shown only by the electrocardiogram, usually indicates a grave prognosis unless syphilis be the etiological factor. Changes in the "T" wave, especially inversion of the "T" wave in Lead I, Leads I and II, or Leads I, II, and III combined, are known to be of serious prognostic significance, providing digitalis has not been recently administered. Ventricular tachycardia and ventricular fibrillation, which can be definitely diagnosed only with the electrocardiograph, are grave conditions, the latter usually being observed only in the dying heart.

VALUE IN TREATMENT

As in prognosis, the value of the electrocardiographic method in treatment of heart disease is still in the academic and developmental stage. The action of certain drugs like digitalis, strophanthin, quinine derivatives, arsenicals, atropine, physostigmine, adrenalin, potassium salts, muscarine, aconitine, and others is being better understood because of careful experimental and clinical electrocardiographic studies. The effect of digitalis on the "T" wave may be studied during treatment to insure the desired therapeutic effects, or the observations may be used as a method of standardizing the drug. Before giving quinidine to restore a fibrillating auricle to a normal rhythm, it is well to know something about the ventricular muscle. If the patient is suffering from extensive myocardial damage and congestive heart failure, it would be unwise to proceed with such treatment until the congestion had been cleared up with rest, depletion, restriction of fluids, and digitalis. It would probably be unwise to employ quinidine if the heart muscle shows extensive damage. The electrocardiogram would be of value in making our final decision about treatment. In heart-block due to syphilis, the progress of the treatment with arsenicals and iodides can be readily shown by electrocardiograms. Patients with heart-block who are taking atropine may have the progress of treatment carefully checked by this method. As time goes on, no doubt other avenues of study with the electrocardiograph in therapeutics will be revealed and older methods will be abandoned as a better clinical understanding is established.

SUMMARY

The electrocardiographic method has served a useful purpose in clearing up much of the mystery concerning disordered mechanisms of the heart. The close parallelism between experimental and clinical observations affords the most brilliant example of applied physiology up to this time.

The electrocardiograph, with such modifications as may be made, will remain an instrument of great value to the investigator. Its permanent place in medical education is assured, for study and research

and in the instruction of successive classes of students.

All medical students should be taught the interpretation of the commoner types of electrocardiograms, and they should be instructed to recognize most of the disordered mechanisms of the heart without the aid of any instrument of precision.

Practitioners who have not had the advantage of instruction in the use of the instrument and what it reveals should make an effort to learn the commoner types of abnormalities. Polygraphic study will be of assistance if an electrocardiograph is not available.

The electrocardiogram serves as a permanent record for comparison and study.

The value of the electrocardiographic method in prognosis and treatment is great, but will not be fully realized for many years to come.

Finally, it should be remembered that the electrocardiographic method is an aid in clinical medicine to confirm the findings of the physicians, and to stimulate him towards improved methods of observation.

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BIBLIOGRAPHY *

1. Ader, C.: Sur un nouvel appareil enregistreur pour cables sous-marine, *Comptes Rendus de l'Acad. des Sc.*, 1897, cxxiv, 1440.
2. Barker, L. F.: Electrocardiography and Phonocardiography, *Bull. Johns Hopkins Hosp.*, 21: 358-389, 1910.
3. Buchanan, J. A.: A Study of the S-T Interval in 1028 Electrocardiograms, *Arch. Int. Med.*, 28: 484-494, 1921.
4. Carter, E. P.: Further Observations on the Aberrant Electrocardiogram Associated With Sclerosis of the Atrioventricular Bundle Branches and Their Terminal Arborizations, *Arch. Int. Med.*, 22: 331-353, 1918.
5. Carter, E. P. and Greene, C. H.: The Electrocardiogram and Ventricular Preponderance, *Arch. Int. Med.*, 24: 638-644, 1919.
6. Coffen, T. H.: The Favorable Prognosis in Auricular Fibrillation, *J. A. M. A.*, 81: 440, 1923.
7. Cohn, A. E., Fraser, F. B., and Jamieson, R. A.: The Influence of Digitalis on the T Wave of the Human Electrocardiogram, *Jour. Exper. Med.*, 21: 593-604, 1915.
8. Cohn, A. E.: Present Status of the Electrocardiographic Method in Medicine, *Am. J. Med. Sc.*, 151: 529, 1916.
9. Cohn, A. E.: A New Electrode for Use in Clinical Electrocardiography, *Arch. Int. Med.*, 26: 105-114, 1920.
10. Cotton, T. F.: Observations on Hypertrophy, *Heart*, 6: 217-226, 1915-1917.
11. Cushny, A. R.: On the Interpretation of Pulse Tracings, *J. Exper. Med.*, 4: 327, 1899.
12. DuBois Reymond (1849), quoted by Satterthwaite.
13. Einthoven, W.: Le telecardiogramme, *Arch. internat. d. physiol.*, 4: 132-165, 1906.
14. Einthoven, W., Flohill, A., and Battaerd, P. J. T. A.: Die Registrierung der menschlichen Herztöne mittels des Saitengalvanometers, *Arch. f. d. des. Physiol.*, 117: 461-472, 1907.
15. Einthoven, W.: The Different Forms of the Human Electrocardiogram and Their Significance, *Lancet*, 1: 853-861, 1912.
16. Einthoven, W., Fahr, G., and de Waart, A.: Über die Richtung und die manifeste Grösse der Potentialschwankungen im menschlichen Herzen und über den Einfluss der Herzlage auf die Form des Elektrokardiogrammes, *Arch. f. d. ges. Physiol.*, 150: 275-315, 1913.
17. Eppinger, H., and Rothberger, C. J.: Zur Analyse des Elektrokardiogrammes, *Wien. Klin. Wchnschr.*, 22: 1091-1098, 1909.
18. Eyster, J. A. E. and Meek, W. J.: The interpretation of the Normal Electrocardiogram. A Critical and Experimental Study, *Arch. Int. Med.*, 11: 204-247, 1913.
19. Eyster, J. A. E. and Evans, J. S.: Sino-auricular Heart-block, With Report of a Case in Man, *Arch. Int. Med.*, 16: 832-845, 1915.
20. Eyster, J. A. E. and Meek, W. J.: Experiments on the Origin and Conduction of the Cardiac Impulse. VI Conduction of the Excitation from the Sino-auricular Node to the Right Auricle and the Auriculoventricular Node, *Arch. Int. Med.*, 18: 775-800, 1916.
21. Frank, O. and Hess, O.: Ueber des Cardiogramm un den ersten Herzton, *Verhandl. d. Kong. f. inn. Med.*, 25: 285-291, 1908.
22. Garrey, W. E.: The Nature of Fibrillary Contraction of the Heart: Its Relation to Tissue Mass and Form. *Am. J. Physiol.*, 33: 397, 1914.
23. Gerhard, D.: Arrhythmia perpetua des Pulses, *Deutsch. med. Wchnschr.*, 1: 448, 1907.
24. Hering, H. E.: Ueber die häufige Kombination von Kammervenenpuls mit Pulsus irregularis perpetuus, *Deutsch. med. Wchnschr.*, 1: 213-215, 1906.
25. Hewlett, A. W. and Wilson, F. N.: Coarse Auricular Fibrillation in Man, *Arch. Int. Med.*, 15: 786-792, 1915.
26. Hirschfelder, A. D.: Diseases of the Heart and Aorta, Philadelphia, Lippincott, 1918.
27. Hoffman, A.: Zur Deutung des Elektrokardiogrammes, *Arch. d. f. ges. Physiol.*, 133: 552-578, 1910.
28. Jolly, W. A. and Ritchie, W. T.: Auricular Flutter and Fibrillation, *Heart*, 2: 177-221, 1910-1911.
29. Kahn, R. H.: Beiträge zur Kenntnis des Elektrokardiogrammes, *Arch. d. f. ges. Physiol.*, 126: 197-224, 1909.
30. Kahn, R. H.: Weitere Beiträge zur Kenntnis des Elektrokardiogrammes, *Arch. f. d. ges. Physiol.*, 129: 291-328, 1909.
31. Kerr, W. J. and Bender, W. L.: Paroxysmal Ventricular Fibrillation With Cardiac Recovery in a Case of Auricular Fibrillation and Complete Heart-block While Under Quinidine Sulphate Therapy, *Heart*, 9: 269, 1922.
32. Von Kölliker, A. and Müller, H.: Nachweis der negativen Schwankung am natürlich sich kontrahierenden Muskel, *Verhandl. d. physik-med. Gesellsch., Würzburg*, 6: 528-533, 1855.
33. Kölliker (1850), quoted by Satterthwaite.
34. Kraus, F. and Nicolai, G.: Ueber das Elektrokardiogramm des gesunden und Kranken menschen, *Leipzig*, 1910.
35. Levine, S. A.: Observations on Sino-auricular Heart-block, *Arch. Int. Med.*, 17: 153-175, 1916.
36. Levine, S. A.: Auricular Fibrillation: Some Clinical Considerations, *Am. Jour. Med. Sc.*, 154: 43-55, 1917.
37. Lewis, Sir T.: Auricular Fibrillation: A Common Clinical Condition, *Brit. Med. Jour.*, 2: 1528, 1909.
38. Lewis, Sir T.: Auricular Fibrillation and Its Relationship to Clinical Irregularity of the Heart, *Heart*, 1: 306-368, 1909-1910.
39. Lewis, Sir T.: Mechanism of the Heart-beat, London, Shaw & Sons, 1911.
40. Lewis, Sir T.: Electrocardiography and Its Importance in the Clinical Examination of Heart Affections, *Brit. Med. Jour.*, 1: 1, 1421-1423; 1479-1482, 2: 65-67, 1912.
41. Lewis, Sir T.: Observations on a Curious and not

* For a more complete bibliography up to 1920, see Lewis, T., The Mechanism and Graphic Registration of the Heart Beat, New York, Hoeber, 1920.

- Uncommon Form of Extreme Acceleration of the Auricle. "Auricular Flutter," *Heart*, 4: 171-216, 1912-1913.
42. Lewis, Thomas. The Mechanism and Graphic Registration of the Heart Beat. New York, Paul B. Hoeber, 1920.
 43. Lewis, Sir T.: Observations on Ventricular Hypertrophy, With Especial Reference to Preponderance of One or Other Chamber, *Heart*, 5: 367-402, 1913-1914.
 44. Lewis, Sir T. and Associates: Observations Upon Flutter and Fibrillation. Parts I to IV. *Heart*, 1918-1920.
 45. Lewis, Thomas: Clinical Electrocardiography, London, Shaw & Sons, 1913.
 46. Lippman (1873), quoted by Satterthwaite.
 47. MacKenzie, Sir J.: Diseases of the Heart, London, Frowde, 1914.
 48. MacKenzie, Sir J.: Principles of Diagnosis and Treatment in Heart Affections, London, Frowde, 1916.
 49. MacKenzie, James: The Future of Medicine, London, Oxford Univ. Press, 1919.
 50. Mann, H.: A Method of Analyzing the Electrocardiogram, *Arch. Int. Med.*, 25: 283-294, 1920.
 51. Matteuci (1843), quoted by Satterthwaite.
 52. Meakins, J.: Prolongation of the "S-T" Interval in the Ventricular Complex as Shown by the Electrocardiograph, *Arch. Int. Med.*, 24: 489-497, 1919.
 53. Mines, G. R.: On Dynamic Equilibrium in the Heart, *Journ. of Physiol.*, 46: 349-383, 1913.
 54. Neuhof, S.: Clinical Cardiology, New York, The Macmillan Co., 1917.
 55. Nicolai, G. F.: Das Elektrokardiogramm bei Dextrocardie und untern Lageveränderungen des Herzens, *Berl. klin. Wchnschr.*, 48: 51-55, 1911.
 56. Pardee, H. E. B.: The Prognosis of Auricular Fibrillation, *Jour. Am. Med. Assn.*, 64: 2057-2060, 1915.
 57. Pardee, H. E. B.: The Determination of Ventricular Preponderance from the Electrocardiogram, *Arch. Int. Med.* 25: 683-692, 1920.
 58. Remak (1850), quoted by Satterthwaite.
 59. Ritchie, W. T.: Further Observations on Auricular Flutter, *Quart. Jour. Med.*, 7: 1-12, 1913-1914.
 60. Ritchie, W. T.: Auricular Flutter, Edinburgh, Green, 1914.
 61. Robinson, G. C. and Bredeck, J. F.: Ventricular Fibrillation in Man With Cardiac Recovery, *Arch. Int. Med.*, 20: 725-738, 1917.
 62. Robinson, G. C.: The Significance of Abnormalities in the Form of the Electrocardiogram, *Arch. Int. Med.*, 24: 422-431, 1919.
 63. Rothberger, C. J. and Winterberg, H.: Vorhoffimmern and Ahythmia perpetua, *Wien, klin. Wchnschr.*, 22: 839-844, 1909.
 64. Rothberger, J. and Winterberg, H.: Ueber die Beziehungen der Herznerven zur Form des Elektrokardiogrammes, *Arch. f. d. ges. Physiol.*, 135: 506-528, 1910.
 65. Samojloff, A.: Weitere Beitrage zur Elektrophysiologie des Herzens, *Arch. f. d. ges. Physiol.*, 135: 417-468, 1910.
 66. Satterthwaite, T. E.: Cardiovascular Diseases, New York, Lemcke and Buechner, 1913.
 67. Smith, F. M.: The Ligation of the Coronary Arteries With Electrocardiographic Study, *Arch. Int. Med.*, 22: 8-27, 1918.
 68. Smith, F. M.: Experimental Observations on the Atypical Q-R-S Waves of the Electrocardiogram of the Dog, *Arch. Int. Med.*, 26: 205-220, 1920.
 69. Waller, A. D. and Reid, E. W.: On the Action of the Excised Mammalian Heart, *Phil. Trans. Roy. Soc. London*, 178: 215-256, 1887.
 70. Waller, A. D.: A Demonstration on Man of the Electromotive Changes Accompanying the Heart's Beat, *Jour. Physiol.*, 8: 229-234, 1887.
 71. Waller, A. D.: The Electrical Action of the Human Heart, *Lancet*, May 24, 1913, 1435.
 72. Weiss, O. and Joachim, G.: Registrierung und Reproduktion menschlicher Hertzöne und Herzgeräusche, *Arch. f. d. ges. Physiol.*, 123: 341-386, 1908.
 73. White, P. D. and Bock, A. V.: Electrocardiographic Evidence of Abnormal Ventricular Preponderance and of Auricular Hypertrophy, *Am. Jour. Med. Sc.*, 156: 17-19, 1918.
 74. White, P. D.: Prognosis in Heart Disease in Relation to Auricular Fibrillation and Alternation of the Pulse, *Am. Jour. Med. Sc.*, 157: 5-7, 1919.
 75. Wiggers, Carl J.: Modern Aspects of the Circulation in Health and Disease, Philadelphia and New York, Lea & Febiger, 1915.
 76. Willius, F. A.: Congenital Dextrocardia, *Am. Jour. Med. Sc.*, 157: 485-492, 1919.
 77. Willius, F. A.: Arborization Block, *Arch. Int. Med.*, 23: 431-440, 1919.
 78. Willius, F. A.: Observations on Negativity of the Final Ventricular T Wave of the Electrocardiogram, *Am. Jour. Med. Sc.*, 160: 844-865, 1920.
 79. Willius, F. A.: Observations on Changes in Form of the Initial Ventricular Complex in Isolated Derivations of the Human Electrocardiogram, *Arch. Int. Med.*, 25: 550-564, 1920.
 80. Willius, F. A.: Auricular Fibrillation and Life Expectancy, *Minn. Med.*, 3: 365-380, 1920.
 81. Willius, F. A.: Angina Pectoris: An Electrocardiographic Study, *Arch. Int. Med.*, 26: 192-224, 1921.
 82. Willius, F. A.: Clinical Electrocardiography, Philadelphia, W. B. Saunders Co., 1922.
 83. Wilson, F. N. and Hermann, G. R.: Bundle Branch Block and Arborization Block, *Arch. Int. Med.*, 26: 153-191, 1920.

Serologic Significance of Streptococci in Arthritis and Allied Conditions—In using active serum, R. Burbank and L. G. Hadjopoulos, New York (*Journal A. M. A.*), have developed a technic for bacterial fixations, in the main of the streptococcus types, whereby various arthritic and rheumatoid conditions may be classified serologically into three major groups: Arthritis reacting to hemolytic streptococci and belonging to the iso-atrophic class. This type in pure form is periarticular. Arthritis reacting similarly to hemolytic streptococci, but of different fixing properties. This type is aniso-atrophic or deformans. Arthritis reacting to streptococci of the S. viridans type and belonging to the osteoarthritic or productive form. The bony change is often a very early manifestation in this group, and is demonstrated by crepitus and by roentgen ray. The majority of arthritic cases that are not arrested or cured early in the course of the disease have a tendency to undergo further changes leading to mixed types. Thus, in an acute exudative periartthritis or in an arthritis deformans, the bony parts gradually may become involved and give rise to a mixed serologic picture. Similarly, in a chronic productive arthritic case, acute tonsillitis, or some other hemolytic infection may engraft a synovial (hemolytic) flare-up on a chronic hypertrophic joint. A fair percentage of arthritic patients, especially those suffering from colitis and chronic constipation, have a marked tendency to effect complement fixation in certain strains of non-hemolytic streptococci isolated from the intestinal tract of similar arthritic cases. This type of organism seems to be a transition form of streptococcus hemolyticus, with certain properties lost through secondary intestinal implantation. Certain pathologic conditions have long been known clinically to be precursors of arthritis. Serologically, we have confirmed these clinical observations. In controlling the value of the test in normal cases, the possibility of the presence of some masked focus of infection must be considered. With this point in mind, the test is not diagnostic for arthritis alone, but is diagnostic of a wide group of acute and chronic infections that give rise to anti-streptococcic bodies in human serums.